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Association Of HSCRCP With Hypertension.

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ABSTRACT

Hypertension is one of the most important risk factors for cardiovascular disease and has become an increasingly important contributor to the global health burden. We conducted a study to determine the significance of hsCRP as an inflammatory marker in hypertension and to assess the association between high sensitivity C-Reactive Protein (hsCRP) and hypertension Patients attending the OPD of General medicine at Sree Balaji Medical college and. A total of 50 patients and 50 healthy control subjects were included in the present study. The level of CRP in the serum sample of total 50 patients and 50 control subjects was estimated by a high sensitivity immunoturbidometric assay. Standard unpaired student's 't' test was used for comparison of hs-CRP levels between hypertensive patients and normotensive control subjects. The mean serum hs- CRP level in hypertensive patients was 2.3 mg/L compared to 0.6 mg/L among normotensive control subjects (P<0.001). This study shows that increased serum hsCRP levels are associated with hypertension. Thus, serum hsCRP estimation can be a potential tool for early identification of individuals at the risk for development of hypertension and eventually CVDs.

Keywords: serum hsCRP, Cardiovascular disease, Hypertension.

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INTRODUCTION

Hypertension is one of the most important risk factors for cardiovascular disease and has become an increasingly important contributor to the global health burden [1, 2]. Hypertension is defined as a blood pressure above 140/90 mmHg, with the proportion climbing far higher in persons > 60 years of age [3, 4]. The risk factors for hypertension are only partly known, and accounts for the some of the deficiencies in current primary prevention strategies and in the design of new drugs for the management of this common condition. Recently, chronic low-grade inflammation has been identified as an integral part in the pathogenesis of vascular disease. Experimental data from atherosclerosis models have confirmed the importance of chronic low-grade inflammation in its development and progression [5, 6]. Indeed, several prospective clinical studies have shown that systemic chronic low-grade inflammation is associated with an increased risk of cardiovascular events and mortality [7-10]. Clinical studies have demonstrated increased numbers of well recognized pro-inflammatory markers such as high sensitive C-reactive protein [hsCRP] in patients with hypertension, even after adjustment for potential confounding factors [11]. Furthermore, elevated hsCRP levels have also been shown to be predictive for the development of hypertension in prehypertensive and normotensive patients [12, 13]. In this context, it is proposed to conduct a study on these factors in the population in our area. Hence, the aim of this study is to find the link between inflammation and hypertension, as evidenced by the level of hsCRP in hypertensive patients.

MATERIALS AND METHODS

Patients attending the OPD of General medicine at Sree Balaji Medical college and Hospital for hypertension management were recruited for this study, with prior written informed consent. A total of 50 patients and 50 healthy control subjects were included in the present study and 3ml of blood samples collected and was estimated by a high sensitivity immunoturbidometric method.

RESULTS AND DISCUSSION

The level of CRP in the serum sample of total 50 patients and 50 control subjects was estimated by a high sensitivity immunoturbidometric assay. Standard unpaired student's 't' test was used for comparison of hs-CRP levels between hypertensive patients and normotensive control subjects. The mean serum hs-CRP level in hypertensive patients was 2.3 mg/L compared to 0.6 mg/L among normotensive control subjects ($P < 0.001$). This study shows that increased serum hsCRP levels are associated with hypertension. Thus serum hsCRP estimation can be a potential tool for early identification of individuals at the risk for development of hypertension and eventually CVDs. On comparison with normotensive control subjects, the hs-CRP levels vary significantly with hypertensives. Our study reveals a graded association between blood pressure and CRP elevation in people with hypertension. The statistical analysis was done by SPSS 16 package. The estimation of hs-CRP level in 50 patients and 50 control subjects, revealed significantly high level of hs-CRP (2.4 mg/L) in hypertensive patients compared to control subjects (hs-CRP 0.6 mg/L, $P < 0.001$). In the present study serum hs-CRP levels were estimated in hypertensive and normotensive control subjects, to evaluate any significant relationship between elevated serum hs-CRP levels and hypertension. Similar association between blood pressure (BP) and CRP levels were reported earlier by Blake et al. There are several potential mechanisms that may account for the observed relationship between blood pressure and CRP levels. Increased blood pressure may promote vascular inflammation by modulation of mechanical stimuli from pulsatile blood flow. Cyclic strain has been shown to increase the expression of soluble intercellular adhesion molecule-1 (sICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) by endothelial cells [14] and also upregulate the secretion of monocyte chemoattractant protein-1 (MCP-1) that promote monocyte adhesion to endothelium.

Furthermore, elevated blood pressure is also known to promote generation of reactive oxygen species (ROS) as evident from a study where a significant correlation was observed between levels of CRP and mononuclear oxidative stress [15]. In the light of present findings and from several other studies we hypothesize that hypertension per se may lead to multiple inflammatory stimuli at the vessel wall which in turn promote the production of a number of pro-inflammatory cytokines such as tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6) and CRP as a defense against injurious factors. Inflammation, common in hypertensives, decreases endothelium dependent relaxation, possibly by decreased capacity of the endothelium to generate vasodilatory factors, particularly nitric oxide (NO) which in turn raises blood

pressure. This is substantiated by several studies which have shown inflammatory markers such as CRP as an independent determinant of endothelium dependent vascular function among patient with coronary heart disease (CHD) and this situation may also exist in patients with hypertension (16). CRP inhibits formation of nitric oxide by endothelial cells which in turn promote vasoconstriction, leukocyte adhesion, platelet activation, oxidation and thrombosis. Moreover, high levels of CRP may upregulate angiotensin receptors and enhance expression of plasminogen activator inhibitor-1 by endothelial cells (17). Both these changes could raise blood pressure and promote atherogenesis.

Our findings are in agreement to that reported by Sesso et al, who also have shown a link between elevated CRP and increased risk of developing hypertension in a cohort study, including people with baseline blood pressure in prehypertensive range. Possible mechanisms for this association being oxidative stress and interaction with adhesion molecules, plasminogen activator inhibitor-1 and low density lipoprotein cholesterol (LDL-C) uptake (18). The findings of the present study, therefore suggest the estimation of CRP levels as an essential / potential tool for early identification of individuals at risk for development of hypertension and eventually cardiovascular diseases.

Glucose, urea and creatinine results naturally show no much significant difference between the cases and controls as the cases are chosen applying the exclusion criteria i.e no diabetes, no renal cases.

In conclusion, the above results suggest that increased serum hsCRP levels are associated with hypertension in people of this area attending Sree Balaji Medical college and Hospital. Thus serum hsCRP estimation can be a potential tool for early identification of individuals at the risk for development of hypertension and eventually CVDs.

Table 1: High Sensitive C Reactive Protein (hsCRP mg/L)

	N	Mean	Std.Deviation	T-value	P-value	Sig
Cases	50	2.39	0.26	37.5	0.000	
Controls	50	0.63	0.19			

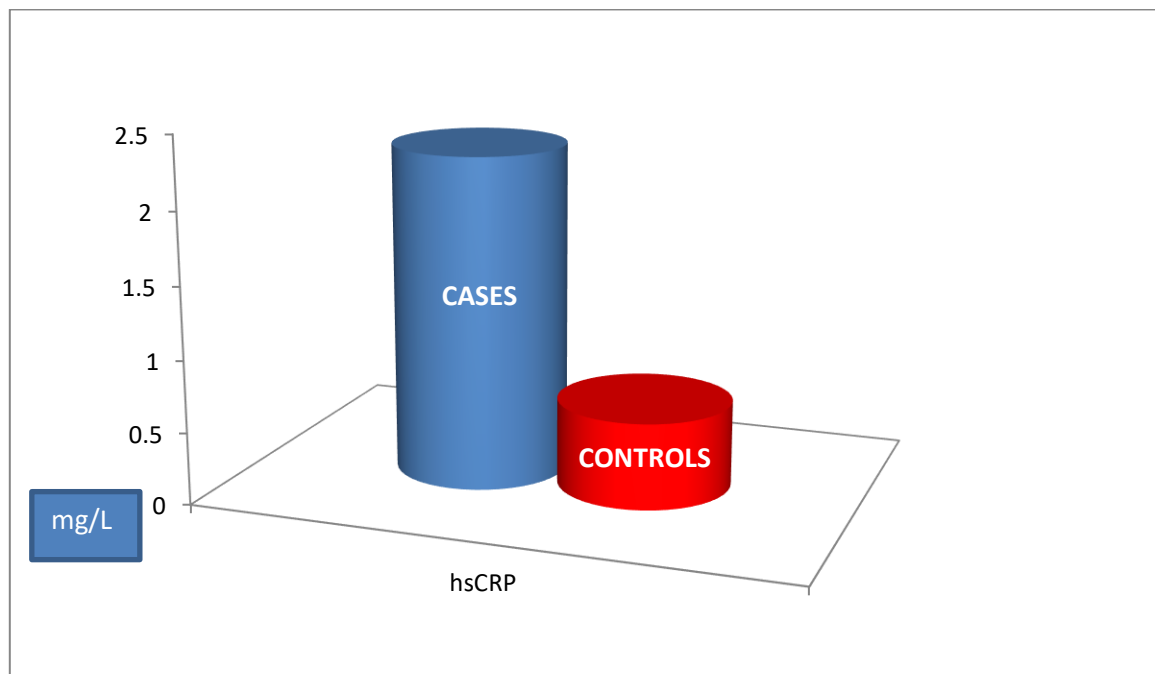


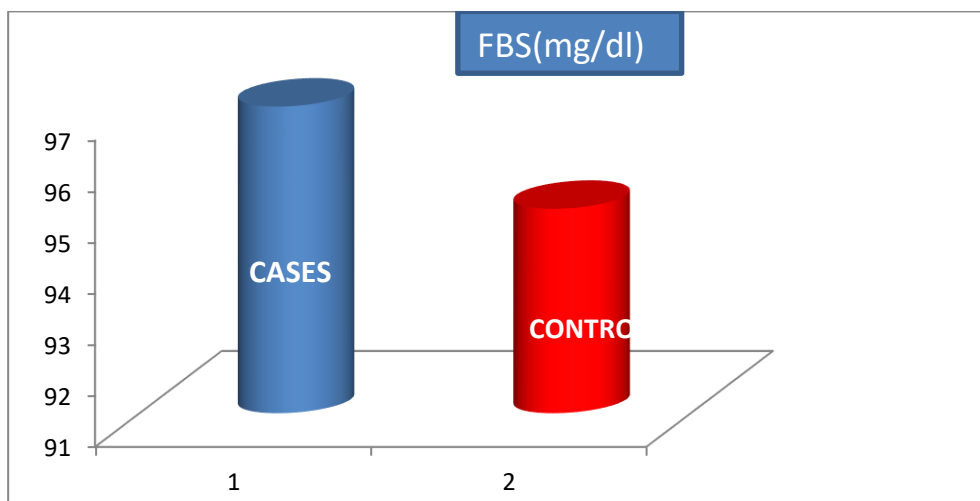
Figure 1: Comparison of hsCRP Levels in Cases and Controls

High sensitive C reactive protein was significantly elevated in hypertensives compared with the controls.

Table 2: FBS

FBS(mg/dl)	MEAN	STD.DEVIATION	T-VALUE	P-VALUE
CASES 50	96.9	14.5	0.8	.424 (NS)
CONTROL 50	95	8.2		

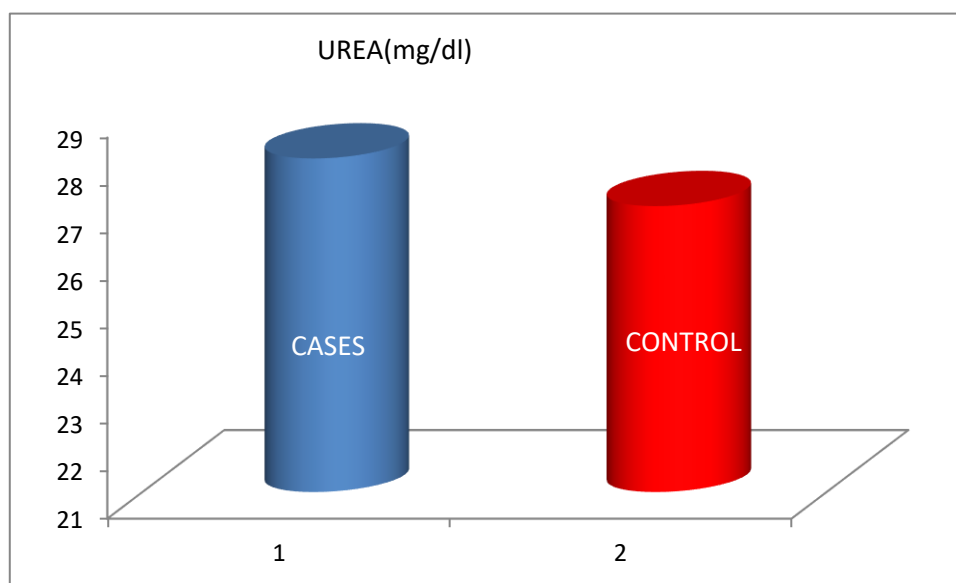
There in no significance in FBS levels of cases as well as controls of the study.



There in no significant difference in FBS levels of cases and controls of the study.

Table 3: Urea (mg/dl)

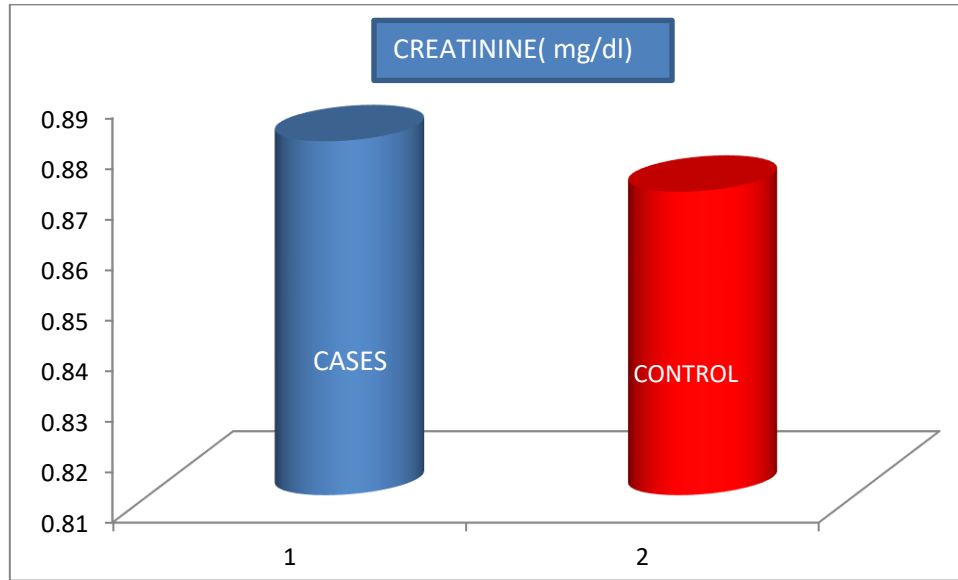
	N	MEAN	STD. DEVIATION	T-VALUE	P-VALUE
CASES	50	28.1	6.5	0.3	0.7
CONTROLS	50	27.7	5.2		



There is no significant difference in urea levels of patients as compared with the controls involved in the study.

Table 4: Creatinine (mg/dl)

	N	MEAN	STD DEVIATION	T-VALUE	P-VALUE
CASES	50	0.88	0.119	37.5	0.50
CONTROL	50	0.87	0.118		



There is no significant difference in creatinine levels of compared with the controls involved in the study.

Table 5: Lipid Profile.

There was no much change in lipid profile of the cases as compared to controls and the values are illustrated below:

Group Statistics

	group	N	Mean	Std. Deviation	Std. Error Mean
TOT CHO	case	50	166.54	30.059	4.251
	control	50	99.76	13.996	1.979
TGL	case	50	157.30	55.730	7.881
	control	50	102.26	19.575	2.768
HDL	case	50	43.10	4.161	.588
	control	50	42.32	8.097	1.145
LDL	case	50	90.98	25.739	3.640
	control	50	33.82	9.169	1.297
VLDL	case	50	32.02	10.215	1.445
	control	50	20.84	4.950	.700
RATIO	case	50	3.854	.8401	.1188
	control	50	2.286	.3574	.0505

CONCLUSION

This study shows that increased serum hsCRP levels are associated with hypertension. Thus serum hsCRP estimation can be a potential tool for early identification of individuals at the risk for development of hypertension and eventually CVDs. However these findings should be confirmed in prospective cohort studies, aimed at elucidating the role of hsCRP in the prediction, diagnosis and management of hypertension

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